We claim:

1. A gene-based method for predicting metastasis in a tumor that exists in both metastatic (M+) and non-metastatic (MO) classes, comprising the steps of:

A. Identifying by expression-profiling of tumor sample cohorts of said M+ and MO classes of said tumor, coupled with permutational statistical analysis to generate a candidate gene list, those genes whose expression differ statistically between said classes of said tumor and that are upregulated in the M+ class and downregulated in the MO class;

B. producing a class-predictive algorithm based upon said predictive genes with a permutational *P* value of <0.05; and

C. applying said algorithm to a candidate tumor to produce a Predictive Strength value that will assign the M+ or MO class to said tumor.

2. The method according to claim 1, wherein said expression profiling is carried out using microarrays of oligonucleotide gene chips.

3. The method according to claim 1, wherein said tumor is a neurotumor.

4. The method according to claim 3, wherein said tumor is a medulloblastoma.

5. The method according to claim 3, wherein said tumor is a glioma.

6. The method according to claim 3, wherein said tumor is a neuroblastoma.

7. The method according to claim 3, wherein said tumor is an ependymoma.

8. The method according to claim 1, wherein said tumor is lung cancer.

9. The method according to claim 1, wherein said tumor is breast cancer.

10. The method according to claim 4, wherein said predictive M+ genes that are up-regulated in said metastatic tumor are found in the group consisting of: invasion and angiogenesis genes, growth factor or cytokine-mediated proliferative genes, signal transduction genes, transcriptional regulatory genes, DNA duplicative genes, and

oncogenesis genes.

11. The method according to claim 4, wherein said predictive upregulated M+ genes and said predictive downregulated MO genes are as listed in listed in Fig. 1.

12. The method according to claim 4, wherein said predictive gene comprises at least one of the M+ gene group consisting of *PDGFRA, FGFR2, IGFBP2, IGFBP7, RAS/MAPK pathway, PDGFA, ITGA4, ITGB5, SPARC, TIMP1, TIE, HOXA4, HOXA7, NTRK3, MYC, CTSC, CTSD, BLM, TPBG and MSH2*, as these genes are defined in the specification.

13. The method according to claim 12, wherein said upregulated predictive M+ gene is the gene for *PDGFRA.*

14. The method according to claim 12, wherein said upregulated predictive M+ gene is a member of the downstream *RAS*/mitogen-activated protein kinase (*MAPK*) signal transduction pathway.

15. The method of claim 13, wherein said *PDGFRA* M+ gene enhances medulloblastoma migration and upregulates at least one member of the *MAPK* group of genes.

16. The method according to claim 1, wherein said algorithm comprises two primary equations:

(1) $$v_i = [\, x_i - (\mu_{Mo} + \mu_{M+}) / 2 \,]$$

wherein vi is the selective vote, xi is the expression level in the tumor sample, and μMO and μM+ are the metastatic classes of reference samples, and wherein said votes are summed in order to obtain total votes for the non-metastatic (V_{Mo}) and metastatic (V_{M+}) classes; and,

(2) $$\text{Prediction Strength} = [\, (V_{Mo} - V_{M+}) / (V_{Mo} + V_{M+}) \,]$$

wherein Prediction Strength values range between 0 and 1.

17. The method according to claim 10, wherein said Prediction Strength is no less than 0.23.

18. A method for inhibiting or reversing *in vivo* metastisis in a M+ class tumor in a subject, comprising the step of administering to said subject an effective amount and for an effective period of time an inhibitor of the upregulation (overexpression) of a gene identified by the method of claim 1 as being associated with said M+ class.

19. The method according to claim 18, wherein said inhibitor is a neutralizing antibody directed against the protein encoded by said upregulated M+ gene.

20. The method according to claim 18, wherein said inhibitor is a chemical inhibitor.

21. The method according to claim 20, wherein said inhibitor is directed against a member of the the metastatic overexpressed gene group consisting of the signal transduction inhibitor STI-571, the *RAS* inhibitor R115777, the MAP2K1/MAP2K2 protein kinase inhibitor U0126, the specific signal transduction inhibitor of PDGFRA STI-571, the phosphoinositide 3-kinase inhibitor wortannin, the *VEGF* inhibitor NM3, the *MAP* kinase inhibitor CC1-779, and the glutathione S-trandferase inhibitor TLK 886 .

22. The method according to claim 21, wherein said inhibitor is the *RAS* inhibitor R115777.

23. The method according to claim 21, wherein said inhibitor is SCH88336.

24. The method according to claim 21, wherein said inhibitor is U0126.

25. The method according to claim 21, wherein said inhibitor is STI-571.